Aberrant AICA Injury During Translabyrinthine Approach

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Aberrant AICA Injury During Translabyrinthine Approach

Ashley M. Bauer, Kristen Angster, Ari D. Schuman, Byron Gregory Thompson, and Steven A. Telian

Objective: To define a complication of the translabyrinthine surgical approach to the posterior fossa related to a rare variant of the anterior inferior cerebellar artery (AICA) that penetrated into the petrous temporal bone.

Patient: A healthy 59-year-old male with a unilateral sporadic vestibular schwannoma.

Intervention: The patient elected to undergo a translabyrinthine approach for resection of a vestibular schwannoma. An aberrant loop of AICA was encountered during the temporal bone dissection within the petrous portion of the temporal bone.

Outcomes: The patient suffered a presumed ischemic insult resulting in a fluctuating ipsilateral facial paresis and atypical postoperative nystagmus.

Results: MRI demonstrated an ischemic lesion in the vascular distribution of the right anterior-inferior cerebellar artery, including the lateral portion of the right cerebellar hemisphere, middle cerebellar peduncle, and bordering the right cranial nerve VII nucleus. His functional recovery was excellent, essentially identical to the anticipated course in an otherwise uncomplicated surgery.

Conclusions: This case highlights the irregular anatomy of the AICA as well as the importance of thorough neurological exams in the postsurgical lateral skull base patient.

Key Words: Aberrant AICA—Complications—Translabyrinthine approach—Vestibular schwannoma.


The anterior inferior cerebellar artery (AICA) is an important posterior fossa vessel supplying the cerebellum and brainstem. It arises from the basilar artery and enters the cerebellopontine angle (CPA) in close proximity to the facial and vestibulocochlear nerves. Within the CPA, it usually bifurcates into rostral and caudal trunks. The former, also called the nerve-related trunk, follows the facial and vestibulocochlear nerves toward the internal auditory meatus (IAM), often traveling between the nerves (1,2). At the IAM, it forms a loop, which then courses back to the brainstem and cerebellum (2).

The course of the AICA is highly variable. Its origin, most commonly from the basilar artery, can occur from the vertebral artery, the posterior inferior cerebellar artery (PICA), or the internal carotid artery (2). In one study, the bifurcation into the rostral and caudal trunks described above occurred in only 59% of cadaveric samples with the remainder existing as a single artery throughout its entire course (33%) or duplicated throughout its course (8%) (2). Multiple radiological studies of the posterior circulation found that approximately 36% of patients had one absent AICA, with its respective territory vascularized via perforators from the contralateral AICA and an expanded territory for the ipsilateral PICA (3,4). When present, the AICA gives off several arteries within the CPA, including the labyrinthine or internal auditory artery (IAA), perforators to the brainstem, and the subarcuate artery. In most cases, the subarcuate artery originates from AICA medial to the internal acoustic meatus (IAM), penetrates through dura posterolateral to the porus acusticus, and enters the petrous temporal bone through the subarcuate fossa, supplying the mastoid air cells in the region of the semicircular canals (5,6).

There are several surgical approaches utilized for the removal of CPA lesions such as vestibular schwannoma, the most common being the translabyrinthine approach. During this operation, a labyrinthectomy is performed, consequently sacrificing hearing while allowing for distal identification and lateral dissection of the facial nerve (6–8). During the deeper exposure of the IAM and posterior fossa dura, the subarcuate artery is routinely encountered as it penetrates the posterior fossa dura and sacrificed. This report details a particularly unique instance of abnormal AICA anatomy encountered during a translabyrinthine approach for a vestibular schwannoma, in which the loop of AICA penetrated the dura and posterior face of the petrous bone. Since this unique
anomaly was not suspected upon preoperative review of the available magnetic resonance imaging or computed tomography of the temporal bone, there were no focused vascular imaging studies obtained.

CASE

A 59-year-old man presented with a 4-year history of progressive right hearing loss and episodic vertigo starting 6 months before presentation. His examination was significant for a positive refixation saccade on head thrust to the right. An audiogram showed a mild sloping to profound sensorineural hearing loss in the right ear, with a speech reception threshold of 30 dB with 88% speech discrimination. His hearing on the left showed a mild sloping to moderate sensorineural hearing loss with a speech reception threshold of 15 dB with 100% speech discrimination.

Magnetic resonance imaging (MRI) showed a 9 × 19 mm enhancing right cerebellopontine angle lesion filling the entire internal auditory canal, consistent with a vestibular schwannoma. The patient was primarily seeking relief of his vestibular symptoms and elected to undergo microsurgical resection using the translabyrinthine approach, with the understanding that the likelihood of hearing preservation was judged to be less than 50% using a retrosigmoid approach. During the extradural approach deep to the bony labyrinth, what appeared to be a dramatic enlargement of the subarcuate artery was encountered during drilling, resulting in brisk arterial bleeding that was readily controlled by bipolar cautery. Owing to suspicion that this may have been a large arteriole arising from AICA instead of the typical subarcuate artery, the dural attachment to this vessel was left intact as the posterior fossa dura was opened. This demonstrated that there was a large vessel loop turning sharply and embedded within the dura in the region of the subarcuate fossa, with the downstream portion of the vessel directed intracranially. The vessel loop was overlaying the tumor until the vessels and attached fragment of dura were mobilized (Fig. 1).

Following surgery, the patient initially had normal facial function documented in the postoperative care unit. Thirty minutes later, he developed a sudden paralysis of his right facial nerve, which was transient and fluctuated in degree over the next several hours, eventually becoming a modified House-Brackmann grade III. He was otherwise neurologically intact. On postoperative day 1, his facial nerve function continued to fluctuate, and his nystagmus pattern was atypical for an acute peripheral vestibulopathy. Instead of the anticipated left-beating nystagmus, he had brisk right-beating nystagmus in primary position and with rightward gaze, which changed to left-beating nystagmus on leftward gaze. There were no other neurological changes. An MRI demonstrated an ischemic lesion in the vascular distribution of the right anterior-inferior cerebellar artery, including the lateral portion of the right cerebellar hemisphere, middle cerebellar peduncle, and bordering the right cranial nerve VII nucleus (Fig. 2). Before discharge, his functional recovery was excellent, identical to the anticipated course in an uncomplicated surgery. Subjectively, his balance improved relative to his preoperative vestibular symptoms. He did not require a prolonged hospital stay. At his first postoperative appointment, his facial nerve function had stabilized at grade III, and his nystagmus had nearly resolved. By 3 months, the facial nerve function had improved to a grade II facial paresis on the right. At 1 year, there was a stable grade II recovery, with persisting reduced brow and oral commissure movement but no synkinesis or spasm. His right lower eyelid had a minor ectropion in repose. MRI demonstrated marked reduction of the abnormal signal in the right cerebellum, with no residual tumor.

FIG. 1. The aberrant loop of right AICA supported in anatomic position by the suction tip on the involved dura is visualized in the cerebellopontine angle, posterior to the tumor mass (asterisk). Its attachment to the dura and the location of cauterization (arrow) during petrous temporal bone dissection are evident. AICA indicates anterior inferior cerebellar artery.
DISCUSSION

There are limited reports in the literature describing the AICA anomaly encountered in this clinical case. One report based on a cadaveric dissection described a unilateral vessel loop, identical to this case, entering the dura and petrous temporal bone posterior to the internal acoustic meatus near the subarcuate fossa (8). Tanriover and Rhoton (5) also described this phenomenon in a cadaveric specimen, and commented that they had observed this anomaly in clinical practice in four patients undergoing acoustic neuroma surgery via the retrosigmoid approach. To our knowledge, this is the first clinical case report of this anatomic variant and related complications due to encountering the vessel loop during extradural dissection of the posterior petrous apex. Retrospectively reviewing the patient’s preoperative imaging, a small loop of vessel can be appreciated posterior to the IAM (Fig. 3A), though any intratemporal extension could not be visualized. There was also a subtle change involving petrous face of the temporal bone on CT imaging thought to be related to this anomaly (Fig. 3B). It is doubtful but unknown whether noninvasive MR or CT angiography would have sufficient resolution to identify this anomaly preoperatively. The most consistent course of the AICA includes a sharp turn as it approaches the IAM, forming a well-defined arterial loop with its convexity directed laterally. After giving off the labyrinthine artery, the AICA travels on to supply the cerebellar cortex below the horizontal cerebellar fissure (9). This case demonstrates that the loop of AICA can be embedded in the dura and penetrate the petrous temporal bone, which likely occurs rarely during development. Even though this aberrant vessel was carefully dissected and moved inferiorly to facilitate the tumor dissection, an AICA infarct on the side of the tumor resulted, presumably from earlier cautery. Had this anomaly been encountered during a retrosigmoid approach, it may have interfered with exposure for tumor dissection, particularly obtaining neurotologic access to the internal auditory canal. Though it may have been possible to mobilize the vessel loop from the temporal bone without injury, doing so may have required drilling of the surrounding bone of the petrous apex, potentially exposing the mastoid air cell system and increasing the risk of a cerebrospinal fluid leak.

Isolated AICA infarcts in the general population are rare, and a relatively uncommon complication following vestibular schwannoma surgery. The AICA can supply several regions of the brainstem and cerebellum—most prominently the lateral pons, middle cerebellar peduncle, and anteroinferior cerebellum. The AICA also supplies the cochlea and semicircular canals via the IAA and the subarcuate artery (10). Clinically, the combination of vertigo with unilateral hearing loss in the context of other cerebellar signs should raise suspicion for an AICA stroke (11). In the classic syndrome, symptoms include vertigo and ipsilateral hearing loss with tinnitus, along with ipsilateral facial nerve palsy, ipsilateral dysmetria, and Horner’s syndrome (12). Other reports have included central ocular motor disturbances, including bidirectional gaze-evoked nystagmus, as observed in this case (13,14).

This report highlights a rare but important developmental anomaly of the AICA potentially complicating access to a cerebellopontine angle tumor, whether using a translabyrinthine or retrosigmoid approach. Although it is difficult to detect with routine preoperative imaging modalities, prominence of the subarcuate foramen should increase the index of suspicion, particularly if it appears to traverse the posterior fossa plate of the bony petrous apex. Preoperative verification may influence the surgical approach selected in this setting, and intraoperative surgical technique would be modified seeking to avoid injury to the vessel loop. In our patient, although the anomaly was not detected preoperatively, the diagnosis of an ischemic stroke was suspected due to the intraoperative findings, the unusual fluctuation of facial function as the stroke was evolving, and bidirectional gaze-evoked nystagmus, highlighting the importance of a thorough neurological examination after neurotologic skull base surgery. In our patient, although the anomaly was not detected preoperatively, the diagnosis of an ischemic stroke was suspected due to the unusual fluctuation of facial function as the stroke was evolving, and bidirectional gaze-evoked nystagmus, highlighting the importance of a thorough neurological examination after neurotologic skull base surgery.
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REFERENCES